REMARKS

This Amendment is in response to the Office Action dated January 24, 2008. The claim amendments are believed to place this patent application in condition for immediate allowance; if any issue remains for resolution the undersigned would appreciate a telephone call to resolve such issue at direct dial telephone number 412-281-3350.

The claims have been amended herewith in large part at the urging of the Examiner. The specification and Abstract have also been amended at the request of the Examiner. Support for the claim 22 amendatory language, "Na+H+/ exchanger (NHX)," may be found, for example, at specification page 2 line 20; support for the recitation of stringent hybridizing conditions in amended claim 32 may be found, for example, at page 13 line 22 of the specification. Aside from identifying support in the specification, the undersigned believes the specification, Abstract and claims amendments are self-explanatory. In view of the extensive amendments and the concreteness of the remaining pending claims, it is believed that the non-prior-art based claim rejections and objections, including those under 35 U.S.C. Section 112, first and second paragraphs, have been resolved.

The specification has been amended to insert the necessary identification of SEQ ID NO: 31 and SEQ ID NO: 32 where necessary. Moreover, a new Sequence Listing is being filed herewith. Pursuant to the requirements of 37 C.F.R. Sections 1.821 et seq., Applicant submits the attached Sequence Listing and computer readable form (NewSequence.txt). The nucleotide and amino acid sequences disclosed in the specification and claims may be found in computer readable form NewSequence.txt as filed herewith and as presented in the paper copy of the Sequence Listing filed concurrently with this Amendment. Applicant hereby certifies that the information recorded in computer readable form (CRF) supplied in the accompanying file NewSequence.txt is identical to the accompanying paper copy of the Sequence Listing. The material presented in the computer readable form and the attached Sequence Listing is not new matter because it presents sequences identical to those disclosed in the specification as originally filed.

Further with respect to SEQ ID NO: 31 and SEQ ID NO: 32, these sequences have been extracted verbatim from the specification and each occurrence of the actual single-letter sequence has been labelled by amendment to the specification herewith. The Sequence

Listing provides the three-letter code of the single-letter amino acids provided in the specification. The Examiner will note that certain sequences referred to on page 5 of the specification, final paragraph, are referred to with designations that are not actual sequences in the specification. Therefore, these sequences have not been included in the Sequence Listing nor has any labelling been made of Figure 3, because although the underlying sequences are alluded to on page 5 these sequences per se do not form a part of the present specification.

The rejection of claims 22-25 and claims 30-33 for asserted anticipation has been rendered moot by the combination of claims 26 and 27 with independent claim 22 and all claims dependent thereon. The asserted anticipation rejection is therefore in condition for withdrawal.

The asserted obviousness rejection is also in condition for withdrawal. The plant growth characteristic of **increased yield/biomass or modified plant architecture,** which results by introducing and expressing the isolated nucleic acid according to SEQ ID NO. 1 (encoding the protein according to SEQ ID NO. 2) is now specifically set forth in the claims. As explained below, nothing like obtaining this claimed plant growth characteristic (particularly under non-salt stress conditions) is taught by the cited art taken alone or in combination.

Fukuda et al. (EP 1143002) and Wu et al. (Plant Cell Physiol. 39, 885-889, 1998) cannot alone or together teach the claimed invention. Fukuka et al. disclose the Na+/H+ antiporter gene, but applicants have uniformly admitted that the Na+/H+ antiporter (exchanger) gene is not new to the present inventors. Wu et al. teach seed-specific promoters which, as shown in the publication, specifically drive expression in the seeds, and the described promoters are active in developing seeds in particular (see p. 887, right-hand column). On the other hand, NHX is a protein that has heretofore ubiquitously been associated with increase of salt stress tolerance, such as described not only by Fukuda et al. EP 1143002 but also in the background section of U.S. Patent No. 6,861,574. Even with the benefit of hindsight, plants themselves would only benefit from increased stress resistance—even as to salt—if NHX were expressed during the plant's active growth phase, not during a later stage such as seed maturation. The Wu et al. disclosure does not discuss using seed-specific promoters for any purpose other than to improve seeds, certainly not to improve or increase yield/biomass or plant architecture in any way. One skilled in the art and consulting Wu et al. would not be led to try to improve any plant part other than seeds and, at most, would possibly consider the combined references as

suggesting increased NHX expression in seeds to improve salt stress tolerance in seeds. One skilled in the art does not learn the surprising utility of increased expression of NHX **in improving plant yield/biomass and/or plant architecture** until he or she consults the present specification.

In addition to the above, EP 1143002 to Fukuda et al. cannot combine with Chan et al. (Plant Molecular Biology 22, 491-506) to teach the claimed invention. Chan et al. does not anywhere disclose that the nos promoter is a weak constitutive promoter. The Chan et al. abstract mentions only that the rice alpha-amylase promoter is active in a number of tissues, but not in all, based on GUS expression. Moreover, on page 494, an assay is described for NPTII activity, but this is not a quantitative assay since the assay is not based on a defined amount of protein (there is no determination of the content of NPTII protein in the leaf samples), so no conclusions can be drawn regarding expression levels of the Pnos promoter. In particular, in Figure 3 of Chan et al. on page 497, a person skilled in the art would deduce that the insertions are single copy insertions (except for T4 (lane 11)). From these data, a person skilled in the art might understand that, for a given specific activity, expression of the NPTII protein would be sufficiently high to overcome the selection by G418. The skilled person would assume that in case of low expression levels, a selection towards multicopy insertions would take place since only multiple copies of the gene would be able to compensate for the low expression levels caused by a weak promoter. In no case does any of the Chan et al. disclosure suggest, even in combination with Fukuda et al. or any other prior art, that the claimed increased yield/biomass or altered plant architecture could result from the introducing and expressing in the plant of the nucleic acid sequence of SEQ ID NO. 1 in the sense orientation and under the control of a promoter selected from the group consisting of a seed-specific promoter and a tissue-specific promoter. Claim 22 in its current form, and all claims dependent thereon, therefore recite multiple inventive features not taught at all in either Fukuda et al. EP 1143002 or Chan et al. or the two in combination. At most it might have been obvious to combine a promoter of Chan et al. with the gene of Fukuda et al. to increase salt stress resistance—but the two references even together cannot in any way direct one skilled in the art to the surprising improvement of the invention whereby the NHX gene can improve yield/biomass or plant architecture regardless of (in the absence of) salt stress.

CONCLUSION

For reasons including those explored above, claims 22, 25, 28-33 and 52-53 are now in condition for allowance. Should any issue remain for resolution prior to allowability, the undersigned respectfully requests a telephone call to her direct dial number, 412-281-3350.

Respectfully submitted,

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